In the Specification:

At page 1, line 10, please insert the following paragraph.

A1

--This invention was made with Government support under Grant No. ROIGM60396-01, awarded by the National Institutes of Health and Grant No. MCB-9506191. The Government has certain rights in this invention.--

Please replace the paragraph beginning at page 22, line 15 with the following:

A2

--Due to the basal regulation of anion and K^+ channels in *abh1* without addition of exogenous ABA, experiments were pursued to analyze whether mechanisms lying further upstream confer ABA hypersensitivity in *abh1*. Anion channels are activated and inward-rectifying K^+ channels are down-regulated by upstream $[Ca^{2+}]_{cyt}$ elevations (J.I. Schroeder & S. Hagiwara, *Nature,* 338:427 (1989)). Therefore we directly investigated whether *abh1* modulates ABA-induced $[Ca^{2+}]_{cyt}$ elevations in time-resolved cameleon $[Ca^{2+}]_{cyt}$ imaging experiments (G. J. Allen *et al.*, *The Plant J.,* 19:735 (1999)). Stomata were opened by exposing plants for 12 hours to 95% humidity. In wild-type, 56 % (n=32 of 57) of guard cells showed no $[Ca^{2+}]_{cyt}$ increase in response to a low concentration of 0.5 μM ABA and the remaining 44% (n=25) cells typically showed only one $[Ca^{2+}]_{cyt}$ increase with an average peak increase of 170 ± 25 nM $[Ca^{2+}]_{cyt}$. Interestingly, in *abh1* guard cells, 0.5 μM ABA elicited $[Ca^{2+}]_{cyt}$ increases in 64% of guard cells (n= 41 of 64 cells) with a larger average peak increase of 280 ± 22 μM. Only 19% of the cells (n=12) responded with one $[Ca^{2+}]_{cyt}$ elevation while 45% of *abh1* cells (n=29) showed multiple repetitive $[Ca^{2+}]_{cyt}$ increases at 0.5 μM ABA. Only 36% of *abh1* cells (n=23) showed no response to 0.5 μM ABA. Statistical analyses of responsive versus non-responsive cells confirmed that the ABA responsiveness of *abh1* guard cells was significantly enhanced (χ^2=4.96, P<0.03). Furthermore both the number of $[Ca^{2+}]_{cyt}$

A2 cancel'd

transients per cell ($P<0.001$) and their amplitudes ($P<0.01$) were significantly larger in *abh1* than in wild-type. $[Ca^{2+}]_{cyt}$ imaging analyses and stomatal aperture measurements demonstrate that the *abh1* mutation enhances early ABA signaling mechanisms upstream of ABA-induced $[Ca^{2+}]_{cyt}$ elevations.

Please replace the paragraph beginning at page 23, line 17 with the following:

A3

The *ABH1* gene was identified by plasmid rescue and the corresponding cDNA (2547 bp) was isolated. Briefly, a 278 bp genomic fragment adjacent to the right border of the T-DNA insertion was isolated from *abh1* plants using plasmid rescue as follows: 5 μg of genomic DNA was digested with *Hind*III, self-ligated and transformed into *E. coli* ElectroMAX DH12S (GibcoBRL, Lifetechnology). Plasmid extracted from cells growing on carbenicilin was sequenced. Primers were then generated to amplify 5316 bp genomic DNA flanking the rescued sequence (GenomeWlkaer Kit, Clontech). A 8248bp *Cla*I genomic fragment containing the full *ABH1* locus was cloned from BAC T10F2 (*Arabidopsis* Biological Research Center) into the plant expression vector pRD400. *ABH1* coding sequences were amplified from an *Arabidopsis* Columbia leaf cDNA library by rapid amplification of cDNA ends (RACE PCR, Marathon cDNA Amplification Kit, Clontech) using the plasmid rescue sequence internal primer (5' GAAGCTCAACTCGTTGCTGGAAAG 3'; SEQ ID NO:4) and its reverse. The total cDNA of 2547 bp was then amplified using *pfu* DNA polymerase (Stratagene), cloned in pMON530 and sequenced. *ABH1* 5' UTR (1250bp) was amplified from genomic DNA by PCR using *pfu* DNA polymerase and subcloned in pCAMBIA1303 (Genbank AF23299) containing a promoterless glucuronidase reporter gene. All sequences amplified by PCR were checked by sequencing (Retrogen, CA).

Please cancel the present informal "SEQUENCE LISTING", pages 30 and 31, and insert therefor the accompanying paper copy of the Sequence Listing, page numbers 1 to 5, at the end of the application.